```
=> L1 and deglycosilation
L12           0 L1 AND DEGLYCOSILATION

=> L1 and CD4bs
L13           7 L1 AND CD4BS

=> L1 and CDi
L14          32 L1 AND CDI

=> glycosylation and L1
L15        3292 GLYCOSYLATION AND L1

=> alter2 and L15
L16        1039 2 AND L15

=> prevent (l) glycosylation
L17         888 PREVENT (L) GLYCOSYLATION

=> L17 and L16
L18          28 L17 AND L16

=> CD4 (W) binding (l) L18
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'BINDING (L) L52'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'BINDING (L) L53'
L19           0 CD4 (W) BINDING (L) L18

=> L18 (L) CCR5
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L52 (L) CCR5'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L53 (L) CCR5'
L20           0 L18 (L) CCR5

=> L18 (l) CXCR4
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L52 (L) CXCR4'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L53 (L) CXCR4'
L21           0 L18 (L) CXCR4

=> D L13 IBIB ABS 1-7
```

```
=> gp120 (l) modification or mutation
L1       511138 GP120 (L) MODIFICATION OR MUTATION

=> 197 and 272 (l) L1
L2            6 197 AND 272 (L) L1

=> 197 and 301 (l) L1
L3            0 197 AND 301 (L) L1

=> 197 and 386 (l) L1
L4            0 197 AND 386 (L) L1

=> 272 and 301 (l) L1
L5            0 272 AND 301 (L) L1

=> 272 and 386 (l) L1
L6            1 272 AND 386 (L) L1

=> 301 and 386 (l) L1
L7            0 301 AND 386 (L) L1

=> 197 and 272 and 301  (L) L1
L8            0 197 AND 272 AND 301 (L) L1

=> 197 and 272 and 386 (l) L1
L9            0 197 AND 272 AND 386 (L) L1

=> 197 and 301 and 386 (l) L1
L10           0 197 AND 301 AND 386 (L) L1

=> 272 and 301 and 386 (l) L1
L11           0 272 AND 301 AND 386 (L) L1

=> D L6 IBIB ABS
```

L13 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:536460 BIOSIS
DOCUMENT NUMBER: PREV200200536460
TITLE: Mutagenic stabilization and/or disruption of a CD4-bound state reveals distinct conformations of the human immunodeficiency virus type 1 gp120 envelope glycoprotein.
AUTHOR(S): Xiang, Shi-Hua; Kwong, Peter D.; Gupta, Rishi; Rizzuto, Carlo D.; Casper, David J.; Wyatt, Richard; Wang, Liping; Hendrickson, Wayne A.; Doyle, Michael L.; Sodroski, Joseph [Reprint author]
CORPORATE SOURCE: Dana-Farber Cancer Institute, 44 Binney St., Jimmy Fund Building, Room 824, Boston, MA, 02115, USA joseph_sodroski@dfci.harvard.edu
SOURCE: Journal of Virology, (October, 2002) Vol. 76, No. 19, pp. 9888-9899. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Oct 2002
Last Updated on STN: 16 Oct 2002

AB The human immunodeficiency virus type 1 (HIV-1) gp120 exterior envelope glycoprotein is conformationally flexible. Upon binding to the host of cell receptor CD4, gp120 assumes a conformation that is recognized by the second receptor, CCR5 and/or CXCR4, and by the CD4-induced (CD4i) antibodies. Guided by the X-ray crystal structure of a gp120-CD4-CD4i antibody complex, we introduced changes into gp120 that were designed to stabilize or disrupt this conformation. One mutant, 375 S/W, in which the tryptophan indole group is predicted to occupy the Phe 43 cavity in the gp120 interior, apparently favors a gp120 conformation closer to that of the CD4-bound state. The 375 S/W mutant was recognized as well as or better than wild-type gp120 by CD4 and CD4i antibodies, and the large decrease in entropy observed when wild-type gp120 bound CD4 was reduced for the 375 S/W mutant. The recognition of the 375 S/W mutant by **CD4BS** antibodies, which are directed against the CD4-binding region of gp120, was markedly reduced compared with that of the wild-type gp120. Compared with the wild-type virus, viruses with the 375 S/W envelope glycoproteins were resistant to neutralization by IgG1b12, a **CD4BS** antibody, were slightly more sensitive to soluble CD4 neutralization and were neutralized more efficiently by the 2G12 antibody. Another mutant, 423 I/P, in which the gp120 bridging sheet was disrupted, did not bind CD4, CCR5, or CD4i antibodies, even though recognition by **CD4BS** antibodies was efficient. These results indicate that **CD4BS** antibodies recognize conformations of gp120 different from that recognized by CD4 and CD4i antibodies.

TITLE: A variable region 3 (V3) **mutation** determines a global neutralization phenotype and CD4-independent infectivity of a human immunodeficiency virus type 1 envelope associated with a broadly cross-reactive, primary virus-neutralizing antibody response.

AUTHOR(S): Zhang, Peng Fei; Bouma, Peter; Park, Eun Ju; Margolick, Joseph B.; Robinson, James E.; Zolla-Pazner, Susan; Flora, Michael N.; Quinnan, Gerald V., Jr. [Reprint author]

CORPORATE SOURCE: Department of Preventive Medicine and Biometrics, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Rd., Bethesda, MD, 20814, USA
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SOURCE: Journal of Virology, (January, 2002) Vol. 76, No. 2, pp. 644-655. print.
CODEN: JOVIAM. ISSN: 0022-538X.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 14 Feb 2002
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AB The human serum human immunodeficiency virus type 1 (HIV-1)-neutralizing serum 2 (HNS2) neutralizes many primary isolates of different clades of HIV-1, and virus expressing envelope from the same donor, clone R2, is neutralized cross-reactively by HIV-immune human sera. The basis for this cross-reactivity was investigated. It was found that a rare **mutation** in the proximal limb of variable region 3 (V3), 313-4 PM, caused virus pseudotyped with the R2 envelope to be highly sensitive to neutralization by monoclonal antibodies (MAbs) directed against conformation-sensitive epitopes at the tip of the V3 loop, such as 19b, and moderately sensitive to MAbs against CD4 binding site (**CD4bs**) and CD4-induced (CD4i) epitopes, soluble CD4 (sCD4), and HNS2. In addition, introduction of this sequence by mutagenesis caused enhanced sensitivity to neutralization by 19b, anti-CD4i MAb, and HNS2 in three other primary HIV-1 envelopes and by anti-**CD4bs** MAb and sCD4 in one of the three. The 313-4 PM sequence also conferred increased infectivity for CD4+ CCR5+ cells and the ability to infect CCR5+ cells upon all of these four and two of these four HIV-1 envelopes, respectively. Neutralization of R2 by HNS2 was substantially inhibited by the cyclized R2 V3 35-mer synthetic peptide. Similarly, the peptide also had some lesser efficacy in blocking neutralization of R2 by other sera or of neutralization of other primary viruses by HNS2. Together, these results indicate that the unusual V3 **mutation** in the R2 clone accounts for its uncommon neutralization sensitivity phenotype and its capacity to mediate CD4-independent infection, both of which could relate to immunogenicity and the neutralizing activity of HNS2. This is also the first primary HIV-1 isolate envelope glycoprotein found to be competent for CD4-independent infection.

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Searches run against the Nucleic Acid Pending database produce two sets of results, with the extensions **.rnpm** and **.rnpn**
Searches run against the Amino Acid Pending database produce two sets of results, with the extensions **.rapm** and **.rapn**

Because they contain data that is confidential, the results of Pending database searches should not be left in the case .